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Identification histopathological characterizations of mercuric chloride toxicity in albino male mice

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Abstract

Mercury compounds have been known as one of most heavy metals which cause adverse effect for both human and animal. Nevertheless, several studies have been documented poison effect of mercury in different aspects, but so far there are few reports related histopathlogical changes are not enough clear according dose-dependent. Therefore, our study focused on histopathlogical examination of kidney and lung tissues which were treated with two different dose of mercuric chloride to elucidate level of toxicity. Thirty male mice have been equally divided in to three groups. First group was treated with distilled water as control and other two groups were given two different doses of mercuric chloride; 1mg\k.g and 4 mg\k.g respectively. All animals have been administrated by intra-peritoneal injection for 60 consecutive days. At the end of experimental; all mice were sacrificed and eviscerated the target organs (kidney and lung) to prepare for histological processing steps. Histopathlogical results have revealed that severity of low dose mercuric chloride administration was minor effect than high dose given in both kidney and lung. That was showing moderate renal structures damages and pulmonary parenchyma destruction. Consequently, these results indicated that toxic activity of mercuric chloride was according to dose-dependent. Subsequently, the moderate damages have been occurred in renal and lung tissues due to long term exposure.

Keywords: Mercuric Chloride, Nephrotoxic, Sub-chronic dose

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دراسة التغيرات النسيجية لكلوريد الزئبق في ذكور الفئران البيضاء

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الخلاصة:

عرفت مركبات الزئبق بأنها واحدة من اهم المعادن الثقيلة التي تسبب تأثيرا ضارا لكل من الإنسان والحيوان. ولذلك، تم اجراء العديد من الدراسات حول التأثير السام للزئبق في جوانبهاالمختلفة، ولكن حتى الآن هناك عدد قليل من البحوث المتعلقة بالتغيرات النسيجية وفقا للجرعة المعتمدة. لذلك، ركزت دراستنا على فحص التغيرات النسيجية من أنسجة الكلى والرئة التي تمت معالجتها مع جرعتين مختلفتين من كلوريد الزئبق لتوضيح مستوى السمية. تم تقسيم ذكورالفئران الثلاثين بالتساوي إلى ثلاث مجموعات. أعطيت المجموعة الأولى ا لماء المقطر ك مجموعة سيطرة، وتم إعطاء مجموعتين أخريين جرعتين مختلفتين من كلوريد الزئبقg.k /mg 1و 4 mg/kg على التوالي. وقد تم اعطاء جميع الحيوانات عن طريق التجريع لمدة 60 يوما متتالية. في نهاية التجريبية. تم التضحية بجميع الفئران وإزالة الاعضاء المستهدفة (الكلى والرئة) للتحضير لخطوات المعالجة النسيجية. وقد كشفت النتائج التقطيع النسيجي وفحص التغيرات النسيجية أن شدة الجرعة المنخفضة لكلوريد الزئبق كانت ذات تأثير ابسط من الجرعة العالية في كل من الكلى والرئة. كان ذلك يظهر الأضرار من خلال تنكس خلاياالكلىوانكماش خلاياالرئة . ونتيجة لذلك، أشارت هذه النتائج إلى أن النشاط السمي للكلوريد الزئبق كان يعتمد على الجرعة. وفي وقت لاحق، وقعت الأضرار في أنسجة الكلى والرئة بسبب التعرض على المدى الطويل.

1- Introduction

Mercury is the third hazard heavy metal in nature, able to causes significant health effects to human and animals through unfavorable pathological and biochemical abnormalities. It occurs naturally in environment, usually in combination with other elements as mercuric compounds or salts. It combines can be organic or inorganic, some of which are soluble in water**(Othman *et al* .,2014).**Mercuric chloride is a most toxic form of inorganic mercury salts having many toxic effects including; nephrotoxicity, hematotoxicity , hepatotoxicity .

Water constitutes a relatively significant source of mercury **(Valko*et al* .,2005).**Mercury poisoning has been reported in human following exposure to mercury and its organic and inorganic derivatives.A form of poisoning is Minamata disease which is a disease of the central nervous system, caused by the consumption of fish neither is it genetically inherited **(Kelly *et al.*, 2006).**

Incidence of mercury poisoning in Iraqhadrecorded in late 1971. Symptoms similar to those seen with Minamata disease affected Japan. The 1971 poisoning was the largest mercury poisoning disaster at its time**(Al-Damluji, 1976).**The widely distribution of mercuric compounds in environment made avoid the exposure to these compounds impossible, as mercury incorporated in products of agriculture, medicine and industrial manufactures **(Sharma *et al* .,2007).**It used as ingredient in dental amalgams and it compounds are found in some drugs, including topical antiseptics, stimulant laxatives, diaper-rash ointment, eye drops, cosmetic products ,and nasal sprays also used in barometer ,manometers ,mercury switches fluorescent lamps thermometers **(parker *et al.*,2004).** The increased mercuric chloride consumption cause disturbances in liver and renal functions. Repeated or prolonged exposure may cause skin sensitization, central nervous system and resulting ataxia, sensory and memory disturbances, tremors, muscle weakness and kidney impairment **(ATSDR ,1998).**In view of above mentioned facts about the ubiquity and toxicity of mercuric chloride present study was aimed to evaluate the toxohistological changesin some internal organ ofwhite miceafter long term consumption of mercuric chloride.

2- Materials and Methods

Thirty (30) whitemice of both sexes (old 8 weeks, weight range 40-50 grams) were obtained commercially and placed in a special housing room at the animal house of College of Veterinary Medicine-Baghdad University.The animals were housed in Polypropylene cages (10 mice /cage) and received water and pelleted food. All mice were kept under controlled conditions of temperature (25ºC) the light-dark cycle was 14-10, **(Hafes, 1970)**.Mercuric chloride powder was obtained commercially and dissolved in distal water for subsequent oral administration. Mice divided equally

into three groups: each with ten adult mice, first group (group A) was orally dosed with mercuric chloride suspension daily at dose (1mg\kg) the second group(group B) dosed orally with (4mg\kg), while the third group (group C) was given distilled water orally throughout the experimental period which was considered a control group. After 60days of treatment with mercuric chloride suspension, mice were sacrificed. Kidney and lung tissue obtained from sacrificed mice and fixed in formalin10% then staining routinely by Hematoxilin and Eosin staining**(Luna , 1968).**

3- Results

In present study, control group showed normal histology architecture figure (1). Mercuric Chloride induced a devastating effects inmice kidneys illustrated by varying degrees of histopathological changes. At low dose1mg\kg (group A) the pathological changes were moderate degeneration of epithelial cells of renal tubules and acidophilic cast formation, figure(2). Also, in other section, amyloid-like substances were observed in addition to necrosis of epithelial cells of renal tubules, figure(3).

On the other hand, the higher dose (4mg/kg) group B showed that the pathological changes were more sever including; necrosis of epithelial cells of renal tubules, atrophy of glomerular tuft and filtrate deposition within Bowmman space and hemorrhage figure(4 and 5).Lung sections of group A showed multiple granulomatous lesions in the interstitial tissue, collapse and emphysema, figure(6).In other sections aggregation of macrophages,lymphocytes and foreigh and langhans giant cells in the interstitial tissue figure(7). However the lung ofmicein the group B showed depletion of bronchial associated lymphoid tissues with vacuolation of epithelial cells of bronchiolesfigure (8).

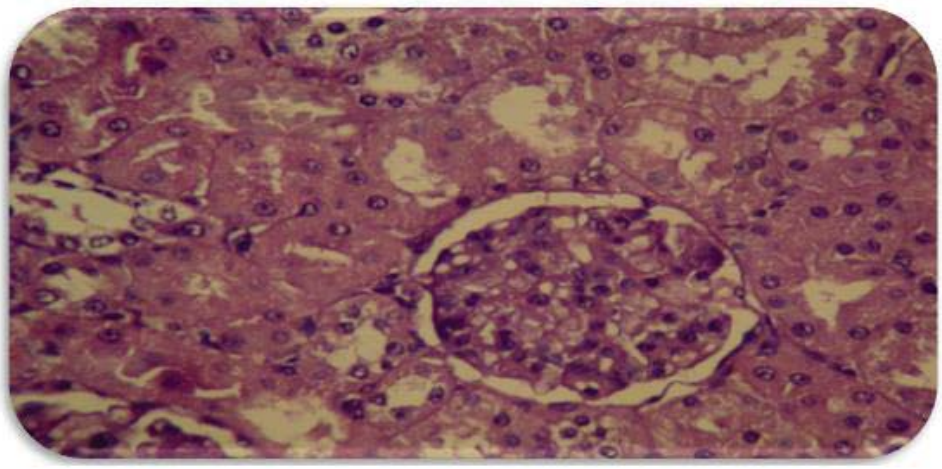

Fig.(1): Histopathological section of untreated mice kidney (control group) shows normalarchitecture (H&E X40)

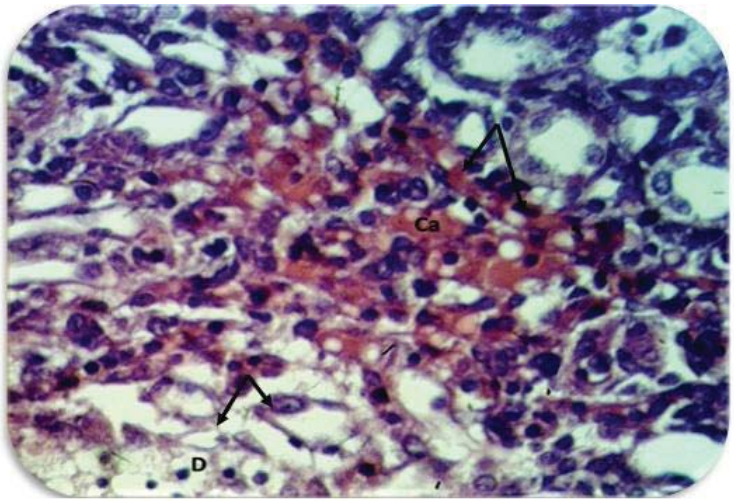

Fig.(2): Histopathological section of kidney of treated (group A) rat shows acidophilic cast (Ca),(D) degeneration of epithelial cells of renal tubules. (H&E 400X)

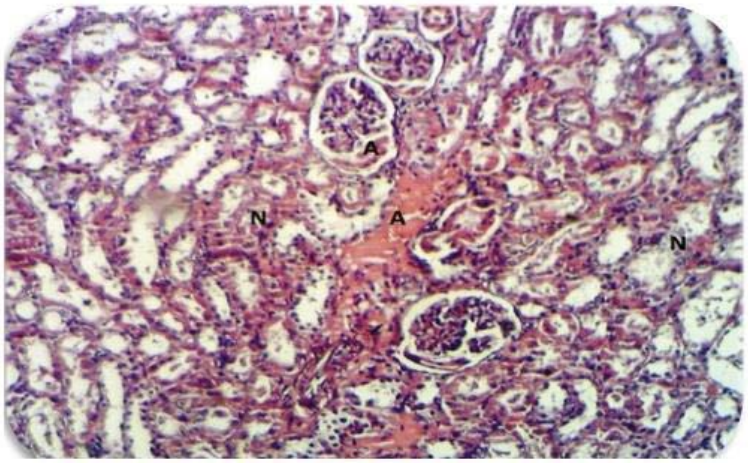

Fig.(3): Histopathological section of kidney of treated(group A) mice shows amyloid deposition (A) and necrosis of epithelial cell of renal tubules (N) H&E 100X

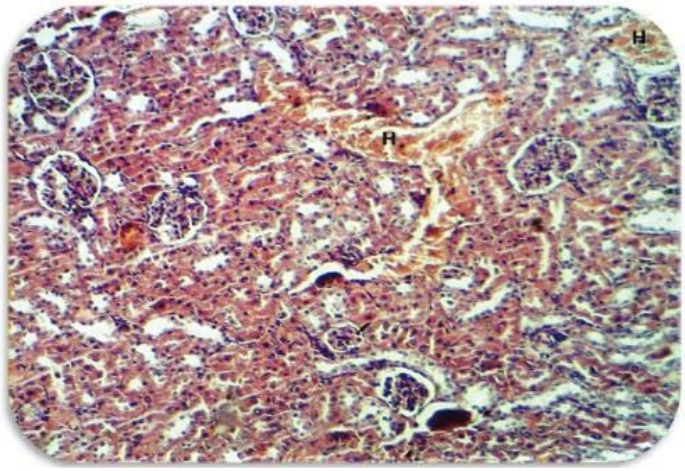

Fig.(4): Histopathological section of kidney of treated(group B) mice shows interstitial hemorrhage and atrophy of glomarulus (H&E X400)

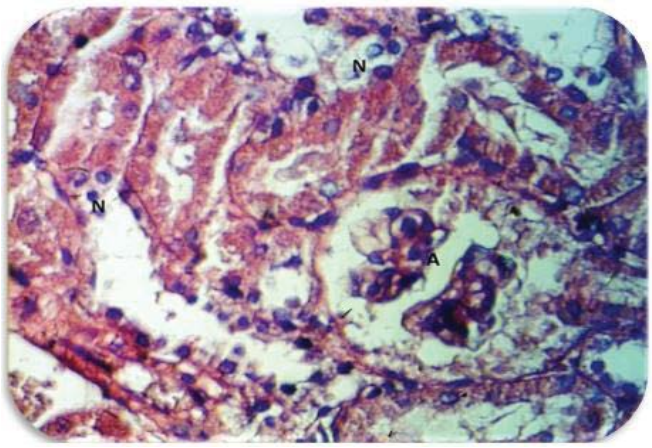

Fig.(5): Histopathological section of kidney of treated (group B) mice shows renal cortex shows tubulonecrosis (N) with atrophy of glomerular tuft (A) and deposition of filtrates within bowman spaces. H&E 400X

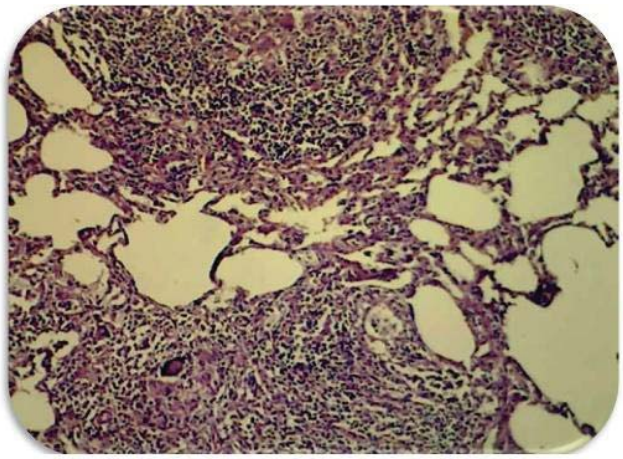

Fig.(6): Histopathological section in the lung of animal 60 days post treatment with 1mg/kg HgCl2 shows multiple granulomatous lesions in the interstitial tissue,collapse and empphysema (H&E stain 40)

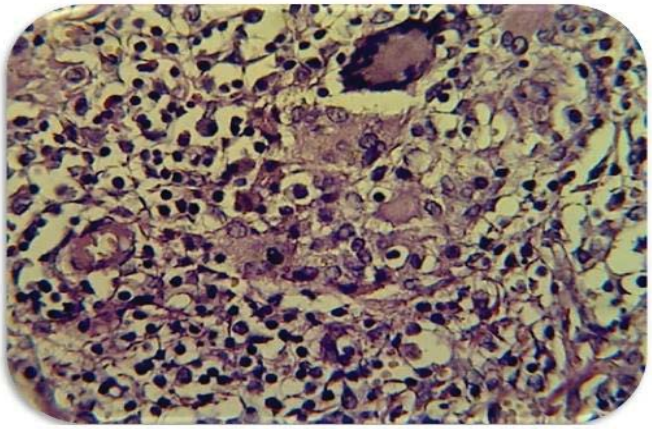

Fig.(7): Histopathological section in the lung of animal at 60 days post treatment with4Hg/kg MgCl2 shows granulomatous lesionsconsisting from aggregation of macrophages,lymphocytes and foreigh and langhans giant cells in the interstitial tissue,(H&E stain 40).

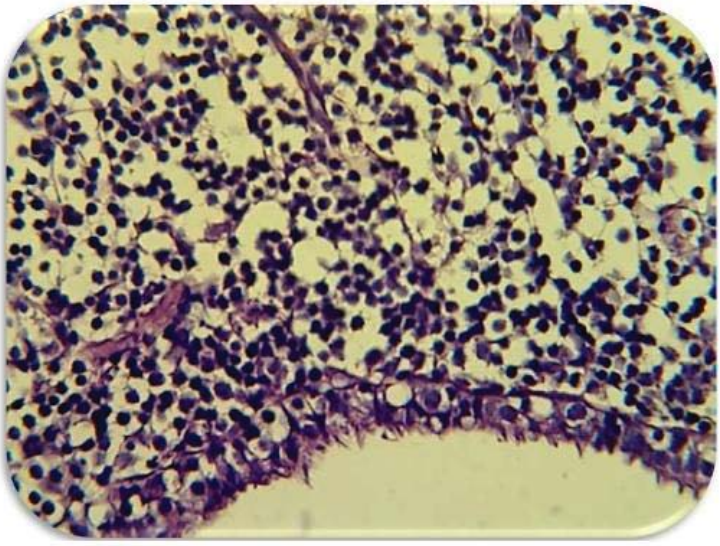

Fig.(8): Histopathological section in the lung of animal at 60days post treatment with 4mg/kg HgCl2 shows depletion of bronchial associated lymphoid tissues with vacuolation of epithelial cells of bronchioles (H&E stain 40)

4- Discussion

Human and animals can exposed to mercuric compounds by two main routs inhalation and ingestion **(Valko*et al* .,2005).** Hence this study undertook the histopathological changes after administration of mercuric chloridesorally. An important result recorded in this study was that mercuric chloride cause tissue damage in kidney and lung at both doses (1mg/kg and 4mg/kg) upon sub-chronic exposure nullifying the suggestion that mercuric chloride are safe at low doses as proposed by **(Kumar *et al* .,2014)**.Moreover, the damaging effect of mercuric chloride was dose-dependent finding which are in agreement with. The renal and lungdamages observed in this study are in agreement with (**Sheikh *et al* .,2011; Ghaleb *et al* .,2012)**. The renal damaged observed in this study could be attributed to high concentration of mercuric chloride accumulated in kidneys, this explanation was depend on the fact that the distribution of mercuric chloride is not uniform in all internal organs in that the major but its accumulated in high concentration in kidney as reported by **(Ghaleb *et al* .,2012; Schiawicke *et al*., 2008).** The mechanism by which mercuric chloride induce renal and lung damage is that after massive accumulation in kidney, the mercuric chloride trigger excessive production of reactive oxygen species and increase lipid peroxidation in the cells **(Chiang, 2001).**

In conclusion mercuric chloride retain toxicity to kindney and lung even at low doses upon long standing exposure, hwoever this toxicity is dose-dependent.

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